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VETENSKAPLIG SKRIFTSERIE

Systematiska kunskapsöversikter 11. Health Impacts of Workplace Heat on Persons with Existing Ill Health

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Innehållsförteckning

Redaktörernas förord

Denna utgåva ingår i den serie av systematiska kunskapssammanställningar som ges ut av Göteborgs Universitet. Dessa kunskapssammanställningar hade sin bakgrund i ett behov att ange riktlinjer för hur man fastställer samband i arbetsskadeförsäkringen. Arbetet inleddes 1981 när en grupp ortopeder, yrkesmedicinare, andra arbetsmiljöforskare och läkare från LO i Läkartidningen diskuterade en modell för bedömning av vilka arbetsställningar som utgjorde skadlig inverkan för besvär i bröst och ländrygg. Gruppen pekade också på vikten av att systematiskt ställa samman kunskap inom området (Andersson 1981). Därefter publicerades flera systematiska kunskapssammanställningar med avsikt ge riktlinjer för förekomst av skadlig inverkan vid arbetsskadebedömningar (Westerholm 1995, 2002, Hansson & Westerholm 2001).

AFA Försäkring finansierar sedan 2008 ett långsiktigt projekt med avsikt att ta fram nya kunskapssammanställningar inom arbetsmiljöområdet. Arbetet samordnas av Arbets- och miljömedicin vid Göteborgs Universitet. Dessa systematiska kunskapssammanställningar har som syfte att beskriva arbetsmiljöns betydelse för uppkomst eller försämring av sjukdom eller symptom i ett bredare perspektiv. Tillämpningen av resultaten får ske inom berörda myndigheter, arbetsplatser och försäkringsbolag.

Kunskapssammanställningarna genomförs av experter inom respektive område. Deras bedömning granskas sedan av andra experter inom området. Den nya serien av systematiska kunskapssammanställningar inleddes 2008 med en förnyad översikt om psykisk arbetsskada (Westerholm 2008), som sedan följdes av sammanställningar om fukt och mögel, helkroppsvibrationer och arbetets betydelse för uppkomst av depression, stroke, Parkinsons sjukdom, ALS, Alzheimers sjukdom och prostatacancer (Torén 2010, Burström 2012, Lundberg 2013, Jakobsson 2013, Gunnarsson 2014, 2015a, 2015b, Knutsson 2017). Under 2016 presenterades ett uppmärksammat dokument om skador efter exponering för handöverförda vibrationer (Nilsson 2016). Dessutom har vi tagit fram ett mycket efterfrågat dokument om hur diabetiker klarar av olika påfrestande arbetsmiljöer (Knutsson 2013). Eftersom kunskapsläget förändras finns det ett behov av uppdateringar av gamla kunskapssammanställningar, samtidigt som det finns ett behov av kunskapssammanställningar inom nya områden.

Detta är den andra systematiska kunskapssammanställningen om betydelsen av exponering för värme. Denna översikt handlar om hur sjuka individer klarar att arbeta i varma miljöer. Arbetet har genomförts av professor Tord Kjellström, Canberra, Australien och forskaren Bruno Lemke, Nelson, Nya Zealand. Externa referenter har varit professorerna Kristina Jakobsson,

Göteborg och Carl-Gustaf Elinder, Karolinska Institutet, Stockholm. Vi är tacksamma för författarnas gedigna arbete liksom de värdefulla och konstruktiva bidrag som referenterna har tillfört.

Göteborg, Lund och Umeå juli 2017

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Maria Albin
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Abbreviations and acronyms

BAU ..... business as usual
(label for doing nothing about climate change mitigation)

CRU..... Climate Research Unit
(at University of East Anglia, Norwich, UK)

GFDL ..... a US climate model that gives values at the low end of all models

GHG ..... greenhouse gases

GTC..... global temperature change

HadGEM ..... a British climate model that gives values at the high end of all models

ILO ..... International Labour Organization

INDC ..... Intended Nationally Determined Contributions
(future GHG emissions from countries)

IOM ..... International Organization for Migration

IPCC..... Intergovernmental Panel on Climate Change

PHS ..... Predicted Heat Strain
(a common heat stress index, physiological)

PIK ..... Potsdam Institute Climate research centre

RCP ..... Representative Concentration Pathways
(scenarios for future global GHG emissions)

TWL ..... Threshold Work Limit
(a proposed occupational health heat stress guidance system)

UNDP..... United Nations Development Program

UNFCCC..... United Nations Framework Convention on Climate Change

UTCI ..... Universal Thermal Climate Index
(new general population heat stress index)

WBGT..... Wet Bulb Globe Temperature
(widely used occupational heat stress index)

WHO ..... World Health Organization

WMO..... World Meteorological Organization

HEALTH IMPACTS OF WORKPLACE HEAT ON PERSONS WITH EXISTING ILL HEALTH

Introduction

Environmental heat stress combines the climatic conditions (temperature, humidity, solar radiation and wind) that impact on the thermal balance of the human body. *Personal heat stress* arises from metabolic activity of muscles for which over 80 % goes into heat production, and the type of clothes worn that may prevent this heat from being lost from the body (Parsons, 2014). *Heat strain* is the adverse response to heat stress leading to loss of productivity, illness and even death. People with specific existing ill health problems are particularly vulnerable to high heat exposures and during physical work the additional health risks are further increased.

The heat risks are likely to be a key problem in hot tropical and sub-tropical areas as the physiological limits to cope with heat exposure are similar in all human beings. However, even in cooler countries, workplace heat can be a problem because much heat comes from inside the body especially during strenuous physical work. A cooler environment only helps the body to lose that extra heat. For example, a study of 361 British military personnel with reported heat illness concluded that 32 % developed heat illness outside the summer months in the temperate British climate (Stacey, et al., 2015). If something like an illness prevents heat loss then there will be disruption to the thermo-regulatory processes and heat strain and heat illness can occur.

Workplace heat and health risks

The issues concerning Occupational Heat Stress have been described in some detail in the report by Kuklane & Gao (2017) in this Arbete och Hälsa series. We will not repeat the descriptions here, but add illustrations and explanations on how people with existing ill health may be particularly affected by heat at work. In summary, the report highlights the heat exchange between the human body and the environment, the role of clothing and metabolic rate (or work intensity), the impacts on heat stress and strain, and the management of heat stress problems. Heat related illness and sick leave occurs in Sweden in spite

of its' cool climate, but most reports come from other countries and in Europe the focus is on heat wave periods. The information and issues mentioned below can be further explored in the Kuklane & Gao (2017) report.

Occupational Heat Stress, and general population heat stress, involves three major components: the "environmental heat", the physical activity (or metabolic heat production) and clothing (Figure 1). The environmental heat in typical work environments, where the surrounding air is the environmental medium for heat exchange, has four major components, which are dominated by air temperature. Air humidity has another important role, as well as air movement (wind speed) and heat radiation. Such radiation emerges primarily from the sun in outdoor work situations, but can also come from workplace specific sources, such as furnaces in steel mills.

The combination of the four components have been expressed in the form of heat stress indices and during the last century more than 170 such indices were proposed (de Freitas & Grigorieva, 2015). Many of these proposed heat stress indices are simplified by not including all four heat components, and most of them have been superseded by improved versions as research and practice has accumulated new evidence. Much heat impact research has just used air temperature as an indicator of heat exposure, but we will refer to WBGT (Wet Bulb Globe Temperature) and UTCI (Universal Thermal Climate Index) as more elaborate indices.

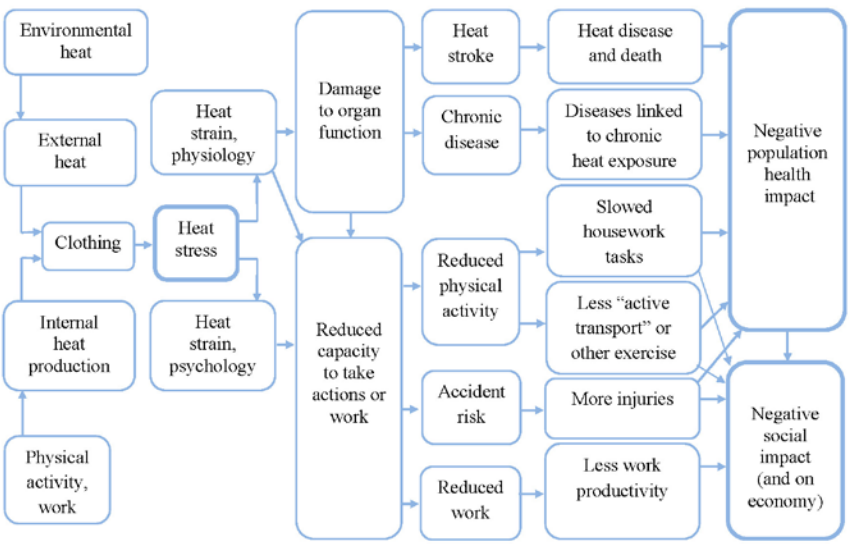


Figure 1. Components and pathways for heat impacts: exposure - stress - strain – disease. (source: modified from Kjellstrom et al., 2016)

WBGT was developed in the 1950s by the US army (Yaglou & Minard, 1957) with the aim of protecting army recruits during training from serious heat effects. WBGT is calculated as a pure environmental heat index (Lemke & Kjellstrom, 2012), but it is linked to interpretation and preventive action guidelines based on the actual experience of heat stress and strain among army recruits during different levels of physical activity and with different clothing. So, it is designed to be applied in work activity situations. WBGT has been criticized (Budd, 2008) as less accurate than heat stress indices based on physiological models, but it was developed from epidemiological data and it is the most widely used workplace heat stress index all across the world.

The UTCI is a recent development and based on a very elaborate physiological model of how environmental heat interacts with the physiological systems of a human body (Fiala et al., 2012). There is limited epidemiological experience of using UTCI in actual work situations and so far its interpretation system is only applied to a light physical activity level and assumptions of standard clothing. Other heat stress indices mentioned by Kuklane & Gao (2017) are Predicted Heat Strain (PHS), Heat Index (HI) and Thermal Work Limit (TWL). PHS calculates predicted sweat rate and body temperature using individual data, and it can be used to assess heat risks based on each persons' sensitivity. HI is a more tentative screening tool for heat stress as it does not take wind speed or heat radiation into account. TWL includes the same components as WBGT, but it incorporates the interpretation scale and calculates the physical activity level (maximum metabolic rate) that is safe in the local environmental heat conditions.

The management of heat stress involves interpretation of the limits for safe core body temperature and accumulated sweat amount. Access to sufficient clean drinking water (and electrolytes/minerals lost during long-term sweating) is a key requirement in any hot work situation. If the accumulated sweat amounts are not compensated by hydration with drinking water (and electrolytes/minerals) serious health impacts may occur already at a few percent loss of body weight. Further details will be given in the text below. The maximum safe body temperature depends on the persons acclimatisation to heat, physical condition and other characteristics (e.g. obesity) (see page 28). If unsafe dehydration or body temperature increase is occurring immediate preventive actions are needed: bring the person to a cooler environment; provide drinking water; reduce work load; etc. If projections of future heat stress would take the person beyond safe limits, preventive management actions need to be included in the work plan.

In heavy labour situations there may be as much as 1 - 1.5 litres of sweating per hour (Baker, 2005). For a full workshift among sugar harvesting workers in Central America, the amount of sweat may be 10 litres or more (Wesseling

et al., 2016). During prolonged sweating the greatest losses of "sweat minerals" is for sodium (one hour of strong sweating may emit more than the full daily intake of sodium) and calcium and iron losses may also be significant (Baker, 2005). During the first hour of intensive work, the main priority to prevent clinical effects of dehydration is to replace liquid lost in sweat. After that, mineral replacements become important and sodium can be provided with salt, sodium chloride (Baker, 2005). It is interesting to note that much of the published research on this topic is based on studies in sports medicine (e.g. Shirreffs et al., 2005; Baker, 2005), while studies of sweating aspects of working people provide few details of the mineral losses. Intensive work in very hot conditions may not be possible to carry out by people with existing ill health, so the extreme dehydration problems may not occur in such people. However, it is worth considering any specific risks in patients with a disease that disturbs mineral balance.

Figure 1 summarizes different elements of heat stress and its impacts on the human body (heat strain) and the eventual impacts on health and well-being. There are both physiological and psychological aspects of the negative effects on heat. Because preventive management actions often involve reduction of the physical workload or taking more and longer rest periods, there is a conflict between protecting health and protecting labour productivity. In the situations when the working person has an existing illness, but is still able to carry out active work, then this conflict may be of particular importance. The well-being of the individual, the family and the community is related to the productivity in the work situation, and heat protection measures that avoid productivity loss would naturally often be of great importance. It should be noted that climate change will increase the health and productivity risks caused by Occupational Heat Stress.

Climate change trends and health threats

Global trends and pathways

The scientific evidence and the awareness of environmental heat as a health hazard has expanded considerably during recent years, even though the basic physiological understanding of the problem has been known for over a century (Parsons, 2014). Climate change and its associated increase of global mean temperature has created new interest in heat as an occupational and general population health hazard (Smith et al., 2014). There is an increasing probability

of people with existing ill health being exposed to excessive heat exposure in their work activities.

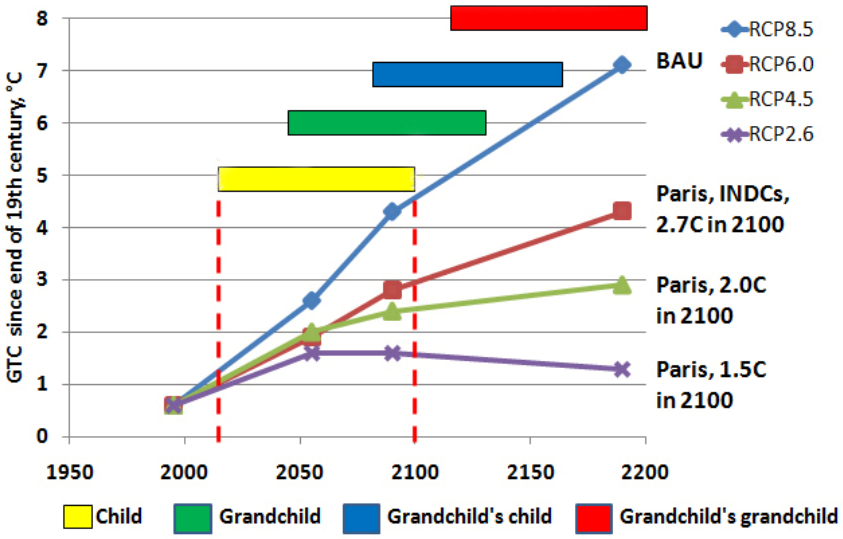


Figure 2. Projected global temperature change trends based on four emission pathways and policies. The likely life span of future generations are indicated. (source: this report)

Figure 2 summarizes a few important issues concerning the projected climate change. The change is often expressed as the Global Temperature Change (GTC) at different time points from now into the future. The starting point of these relationships (the baseline, or historical point) can be 1995 as shown in Figure 2. This is a 30-year average (1981-2010) of GTC since global industrial development expanded in the 19th century. The baseline shown here is 0.74 °C as estimated by the IPCC (Collins et al., 2013). The future trends are calculated with different models (as many as 25 models used by IPCC) and different Representative Concentration Pathways (RCPs).

Figure 2 shows the average GTC trends for all IPCC models (Collins et al., 2013), for the four agreed RCPs. RCP8.5 is the pathway with no actions to reduce climate change (also labelled Business As Usual, BAU). It would lead to a GTC of more than 4 °C at the end of this century, and continuing to almost double by 2200. This will cause very serious global population health consequences (Smith et al., 2014).

The three trend curves below BAU shows the estimated mean GTC under three greenhouse gas emission alternatives approved by the UNFCCC climate change policy conference in Paris in November 2015 (UNFCCC, 2015): INDCs (GTC = 2.7 °C); Paris report maximum (GTC = 2 °C); Paris report ideal (GTC = 1.5 °C). These three GTC levels are very similar to what would

occur with the pathways RCP6.0, RCP4.5 and RCP2.6 (as shown in Figure 2). The Intended Nationally Determined Contributions (INDCs) are the reductions of greenhouse gas emissions individual countries offered before the Paris meeting 2015. If the 195 countries that signed the Paris agreement (UNFCCC, 2015) actually implemented their Intended Contributions the result would be a GTC at 2.7 °C, which is a much higher GTC than needed for protection of global public health (Smith et al., 2014).

Figure 2 also shows that by the middle of this century the GTC is estimated to have reached just below 2 °C regardless of which of the three lower RCPs is implemented, but after that the trends diverge. The two higher RCPs will increase GTC and this increase is expected to continue in the following century, so the health threats will increase beyond the estimates for 2100. It is interesting to note that a child born now has a life span that will most likely last until the end of this century (yellow bar in Figure 2). Many of the people who will be affected by the future climate change late this century are already alive. Figure 2 also indicates the life span for this child's child and two following generations. The grandchild's grandchild can be considered a part of our family, and this person may be alive at the end of the 22nd century. It puts the problems of climate change into a new perspective. As the future climate change and effects on our future family members are dependent on our greenhouse gas emissions now, the need to act for mitigation (reduction) of climate change without further delay is clear (see section 10 for further details).

Future projected trends in workplace heat due to climate change

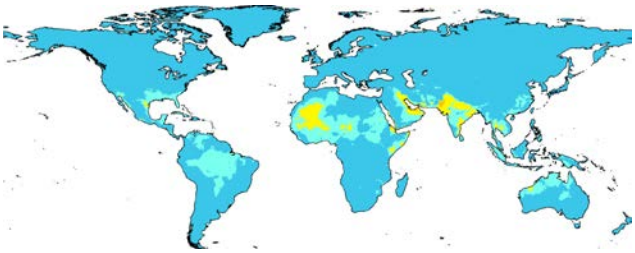
A number of reports from the IPCC have quantified the likely future trends of temperatures, humidity, wind patterns, and other climate variables (e.g. Collins et al., 2013) and the data can be used to calculate spatial and temporal distributions of daily, weekly or monthly heat levels. Kjellstrom et al. (2009) produced the first global estimates and maps of WBGT heat levels, and subsequent reports by this author have given further examples (e.g. Kjellstrom et al., 2016). Figure 3 shows the results of recent analysis of WBGTmax (afternoon values in the shade) in the current situation and at the end of this century.

The current monthly WBGT heat levels in the afternoons during the hottest month are below 28 °C in most of the world (Map 1, Figure 3). This is a level at which moderate labour starts to be affected by the heat and people carrying out less intensive labour will be affected when the levels reach 30 or higher °C (yellow colour in maps). Many of the effects of heat at work are quite acute and therefore will occur even with one or a few days high heat exposure. A monthly average is therefore a crude estimate of the relevant exposure. Map 2 (Figure 3) shows estimates for the lower limit of the 3 hottest days in the hottest

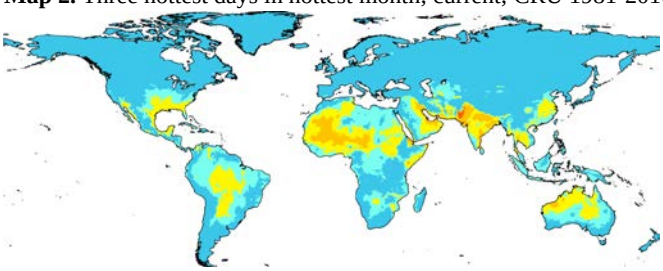
month. This map shows many more hot areas and large areas of the USA and China are now in the yellow range. Almost all of India is yellow or orange.

Figure 3. WBGTmax (afternoon values in the shade) in 67,420 grid cells over land (0.5 x 0.5 spatial degrees). Baseline, current, data (1981-2010) from CRU and future data (2071-2099) from PIK. Method described in detail on website: www.ClimateCHIP.org (in progress). (source: this report)

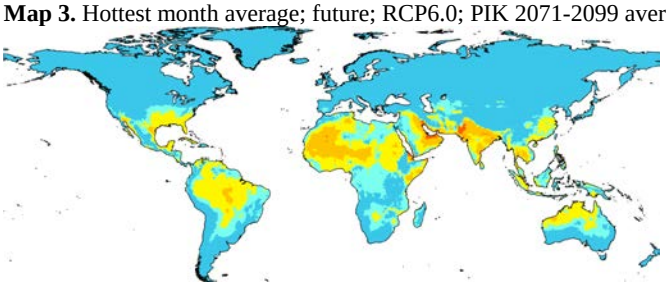
Map 1. Hottest month average; current; CRU 1981-2010 average.



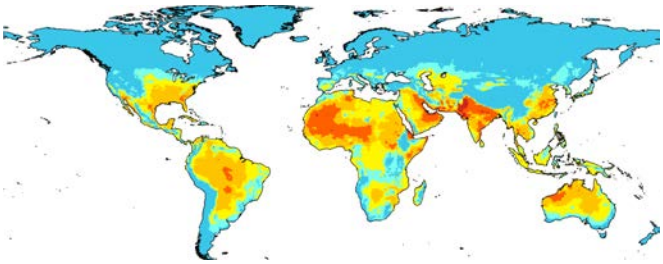
Map 2. Three hottest days in hottest month; current; CRU 1981-2010 average.



Map 3. Hottest month average; future; RCP6.0; PIK 2071-2099 average.



Map 4. Three hottest days; future; RCP6.0; PIK 2071-2099 average.



Europe is still not in the yellow area, but during extreme heat waves and in hot urban locations problems with high WBGT levels will occur there too. The climate models do not take the Urban Heat Island into account, so many urban areas may in fact be 2-3 °C hotter than the levels shown in these maps (Rizwan et al., 2008). In addition, heat exposure while working in the sun is approximately 3 degrees hotter than the levels in the shade shown on the maps.

The future WBGT levels (Maps 3 and 4) are based on the midpoint of the HadGEM and GFDL models and indicate how the hot areas will expand at the end of the century based on RCP6.0 future pathways for GHG emissions. The monthly averages in the future (Map 3) are similar to the 3 hottest days currently, and the 3 hottest days in the future being yellow or orange may have expanded to most of the USA and China. Also large parts of southern Europe are now at this heat level for the 3 hottest days.

Estimates of the number of affected work hours can be made from these heat maps and a recent global report (UNDP, 2016) shows estimates of the annual lost work hours due to heat in moderate intensity work. For some countries the current levels are at a few percent loss, but this will increase to over 10 % in, for instance, Cambodia. The calculation methods are currently being updated to make more precise calculations for countries or local grid cells (see www.ClimateCHIP.org; in progress) and the estimates of current heat and future trends can guide analysis of heat stress hazards for people with existing ill health problems.

It should be noted also that the tropical and sub-tropical areas with the highest heat levels on these Maps are also the areas of the world with the highest population densities. Approximately 2/3 of the global population live and work in these hot areas, and with the ageing populations and expanding groups of people with existing illnesses who need to keep working, the health challenges mentioned in this report will become more and more common. Exact estimates are not available, but one can predict that many million people in these hot areas will experience the combined impacts of heat, work activities and an existing ill health issue.

Summary of health threats due to heat

Heat exposure is only one of the health hazards developing with climate change (Kjellstrom & McMichael, 2013), but it will affect a large proportion of the global population and there are a number of ill health outcomes (Table 1). Logically, the health risks would be increased if a person already have an ill health problem in organ systems affected.

The list in Table 1 includes diseases associated with heat exposure that have not been mentioned in IPCC health impact assessments (Smith et al., 2014), WHO reports (e.g. WHO, 2014), or other major publications. One can assume that the strong link to heart strain and to kidney disease makes it likely that patients with heart and kidney disease are vulnerable to heat exposure in their workplaces. We will explore these additional risks in a later section. It should also be noted that any existing chronic disease is likely to be affected by heat exposure, especially during work, as the physical well-being is reduced by the heat (Kjellstrom et al., 2010).

Table 1. Climate change related health impacts of heat according to IPCC, WHO and other references (source: modified from de Blois et al., 2015 and Kjellstrom et al., 2016)

Hazard exposure	Health impact	Confidence* of this impact	Specific effects at organ level	Source
Intense heat	Heat stroke death	Very high	Heart strain; CNS malfunction; dehydration	IPCC (2014)* WHO (2014)**
	Heat stroke morbidity	Very high	Heart strain; CNS malfunction; dehydration	IPCC (2014)
	Heat exhaustion, work capacity loss	High	Heart strain; mental fatigue	IPCC (2014)
Forced migration	Under-nutrition; infections; mental stress; injuries	High	Work capacity loss, heart strain, mental fatigue	IPCC (2014)

Health concerns not mentioned in detail in the IPCC or WHO reports

Intense heat	Chronic kidney disease linked to dehydration	Wesseling et al., 2016
	Increased incidence of violent crimes	Gamble and Hess, 2012; Raleigh et al., 2014
	Increased incidence of suicides	Berry et al., 2010; Kim et al., 2015
	Teratogenic effects of high body temperature in pregnant women; damage to development of fetal heart and brain	Edwards et al., 1995
	Interactions with prescription drugs	Vanakoski and Seppala, 1998
	Deteriorated clinical status in chronic NCDs	Kjellstrom et al., 2010; Parsons, 2014

CNS = Central Nervous System
* IPCC assessment judgement of the confidence for climate change impact
** the WHO (2014) report is a report on climate change related mortality

Physiology and pathology of heat illness

To understand the interaction of pre-existing illness with exposure to heat, it is important to summarise body processes that occur during heat strain. More detailed descriptions are available in an extensive article on the pathology of heat related illness (Leon & Kenefick, 2011) and a detailed review of heat stroke (Bouchama & Knochel, 2002).

As the core temperature rises to 1 °C above normal, the body's thermos-regulatory systems are activated with the skin blood vessels dilating and the initiation of sweating from sweat glands. The blood is cooled by shunting to the cooler skin, which is kept cool by the evaporation of perspiration (sweat). Vasodilation can increase blood flow to the skin from a normal 0.3L per minute

to 8L per min (Crandall & González-Alonso, 2010). This extra vascular volume drops the blood pressure which stimulates baroreceptor-reflexes to increase the heart rate. If this thermoregulation response is insufficient to compensate for the increase in body temperature then signs and symptoms of heat exhausttion appear including low blood pressure, rapid heart rate, fatigue, dizziness, headache and nausea (Leon & Kenefick, 2011). With increasing age the skin blood flow regulation becomes less efficient (Holowatz et al., 2007, 2010).

To compensate for the increased blood flow to the skin, there is less blood flow to organs especially the intestines and kidneys. Dehydration and salt depletion can occur (from excess sweating).

A number of things happen at the cellular level. A variety of local acting immune system hormones called cytokines are released. These have many actions including mediating fever, stimulating the hypothalamus and modulating local and systemic acute inflammatory response. A further effect is the production of heat shock proteins that increase in conditions of high body temperature or ischaemia. Heat shock protein are “chaperone” molecules that interact with enzymes (and other proteins) preventing them from denaturing (Locke, 1997; Kregel, 2002).

If the person is not cooled quickly the initial heat effects can progress rapidly to heat stroke. Heat stroke is life threatening with central nervous system symptoms such as delirium, agitation, stupor, seizures, or coma (O'Brien, et al., 2012). At this stage the core temperature is usually greater than 40 °C. However, this is not a definitive indicator as core temperature can be above 40 °C without symptoms of heat stroke.

It is now believed that the damage from heat stroke is caused by the systemic inflammatory response syndrome from heat-induced damage to the intestines and other organs (Bouchama & Knochel, 2002). Normally the tight junctions of the intestinal epithelial membrane prevent endotoxin from gut bacteria leaking into the blood. The tight junctions can become “leaky” at higher temperatures or in response to the ischaemia from reduced intestinal blood flow (Hakre, et al., 2004; Barberio, et al., 2015). This “leaky gut” syndrome allows the endotoxins to leak into the blood causing a systemic inflammatory response. The endotoxins may also trigger disseminated intravascular coagulation which is a common clinical symptom of heatstroke. Clots form throughout the cardiovascular system that block arterioles and capillaries and can cause stroke and multi-organ failure.

The main damage in heat stroke is to the brain, liver and kidneys with over 30 % of heat stroke survivors experiencing neurological impairments continuing after initial treatment (Grogan and Hopkins, 2002). Irreversible neuronal damage starts at 40 °C and gets worse as core temperature increases (Kiyatkin, 2010). Hyperthermia also increases the blood–brain-barrier permeability allo-

wing entry of potentially toxic molecules. Liver failure is one of the most common causes of morbidity in patients during the later stages of recovery (24 to 48 hours after exposure). Rhabdomyolysis is caused by injury to skeletal muscles during exertion at high temperatures (Bagley, et al., 2007). Here muscle cell contents (including myoglobin) leaks into the circulation. The myoglobin is toxic to nephrons and causes overproduction of uric acid, which precipitates in the kidney tubules to cause acute renal failure (Bagley, et al., 2007).

If the core temperature is rapidly reduced, seizures controlled and hydration restored, then there is a 90-95 % survival rate of heat stroke victims (O'Brien, et al., 2012). There is a poor prognosis if the core temperature increases to above 41 °C and the hyperthermia is for an extended period. Then, there is acute renal failure, hyperkalaemia and liver function damage (O'Brien, et al., 2012).

Unfortunately action for a heat stroke casualty is usually not that swift and currently this condition is easier to prevent than treat (Bouchama & Knochel, 2002). Preventative measures include: acclimatization to the heat, reduction in the duration and extent of physical activity, rescheduling of activities to cooler times of the day, increased consumption of water, and replacement of lost electrolytes in the sweat after long-term sweating. However, despite these measures there are still people who are vulnerable to heat stress and this article is about the identification of the more vulnerable people.

Acclimatization is a very effective preventative measure. This can occur naturally or can be artificially induced when it is called acclimation. This entails repeated sessions of heat exposure of sufficient intensity to raise core and skin temperature and induce profuse sweating. With daily heat exposure, acclimation takes about 10 days, longer for less frequent exposures (Bouchama & Knochel, 2002). Continual 24-hour exposures are not needed and daily 100-minute exposures were sufficient for acclimatiion in dry heat (Leon & Kenefick, 2011). However continued heat exposures are required to maintain the acclimated state which fades at the same rate as it develops. Lack of acclimatization causes more heat illnesses early in the summer, and could well be an issue for people living and working predominantly in air conditioned premises. It is important to note that acclimatization only gives limited protecttion to heat stress (Périard, et al., 2016) and that people in hot places without access to air cooling systems are mostly already fully acclimatised.

The region of origin also has an effect on a person’s tolerance to heat, with people moving from cooler regions to hotter regions to work being the most vulnerable (Carter, et al., 2005).

Maintaining full hydration has been shown to be one of the most significant factors in the prevention of heat related illness (Périard, et al., 2016). People susceptible to dehydration include those with a weak thirst response (elderly), those who sweat excessively (non-acclimatised workers, especially those

working in high humidity), those unwilling to drink because of conditions such as incontinence or lack of toilet facilities (elderly, women), those with the suppression of hormones that enhance thirst and those who take diuretics that enhance urination. As mentioned earlier, excessive multi-hour sweating due to intensive work in hot conditions can create significant mineral losses (particularly sodium in salt; Baker, 2005) and replacement of lost minerals needs to be considered in heat impact prevention programs.

Humans (and animals) have behavioural responses to heat stress such as reducing activity when it gets too hot (Parsons, 2014). However these psychological responses can be easily suppressed by the determination to complete a task or the inconvenience of stopping. Older people have been known to refuse to turn on air conditioning systems or to leave hot homes during heat waves. Workers may drink insufficient water because they need to carry it to their work place, and piece work employees may continue through the hottest part of the day to make enough money to support their family (MMWR, 2008).

Two types of heat strain

There are two types of heat strain that can occur at the work place: *classic heat strain* and *exertional heat strain*, although the conditions may cause these to overlap (Parsons, 2014). Classic heat strain (also called *non-exertional heat strain*) is when the core body temperature rises to the point where it effects the brain and other organs. The maximum sustainable temperature is about 41 °C, but fatigue and illness can occur at a much lower temperature (38.5 °C) for susceptible individuals. Classic heat strain occurs on exposure to high temperatures if the person is unable to respond to the heat stress appropriately. For example, the inability to recognise the rise in core temperature, so not taking off the clothes that are preventing heat loss. Or carrying out limited physical activities (e.g. physical work) in conditions of high humidity, and not recognising that the humidity is very high. The classic form of heat stress is more prevalent in the very young or elderly especially if they live in hot places and lack air conditioning.

Exertional heat strain is a health threat for working people and people carrying out major physical activity in sports (Parsons, 2014). In this situation the main heat source is the muscle work. Then the blood vessels in the muscles are dilated (for maximum delivery of oxygenated blood), also the skin blood vessels become fully dilated to dissipate the extra heat. This causes a drop in blood pressure leading to an increase in heart rate, and if this fails to restore the blood pressure, there is a reduced blood flow, especially to the internal organs. The resulting ischaemia can lead to multiple organ failure as indicated in Table 2.

Regulation of blood pressure and temperature is vital for life. If blood pressure is too low, then there is insufficient perfusion of oxygenated blood in capillaries to organs especially those that are contracting (muscles, heart, intestines). Insufficient oxygenated blood leads to anaerobic metabolism, ischaemia, inflammation and tissue damage (Leon & Kenefick, 2011).

Table 2. Comparison of symptoms and organs involved in the two types of heat strain. (source: this report)

Heat strain type	Age	Weather	Activity	Sweat	Renal Failure	Rhabdo-myolysis	Hyper-kalaemia	Hypo-glycaemia
Classic	Young, Elderly	Heat-waves	Sedentary	Absent	Not common	Not common	Not common	Not common
Exertional	15-64 yrs	Hot weather	Strenuous Exercise	Profuse	Common	Common	Common	Common

Epidemiological studies of vulnerability to heat exposure

Data for this report were drawn from a number of sources: Increased hospital admissions during and after heat waves (Chicago, France, India, China, Mexico, Australia, Moscow, Western USA), extensive military data, especially from the USA military (USDAAF, 2003), as they strive to lower the heat related deaths during intensive physical training, laboratory climate chamber studies where individual parameters could be controlled, and field studies on working people. More recently monitoring of people participating in sports events such as marathons and football has produced data that would be unobtainable in the controlled conditions of a laboratory, because physiological responses would be out of bounds on what would be approved ethically. Many of the studies were in cities that have a significant "urban heat island" climate where the temperature may be 2-3 °C higher than the surrounding countryside and the local airport weather station (Rizwan, et al., 2008).

Heat waves during recent decades when extensive health impact data has been collected include:

- **Adelaide Australia (1993-2006)**. People with renal disease and with mental health conditions (e.g. senility) were particularly vulnerable. (Li, et al., 2015).
- **Brisbane (1996-2005)**. People with Cardiovascular, Respiratory and renal disease were found to be most vulnerable. (Wang, et al., 2012).

- **Mexico (1998-2002)**. Death rates higher in women than men when usually men are most vulnerable (as they are more likely to get exertional heat illness). (Bell, et al., 2008)
- **Chicago heat wave (1999)**. People with ischaemic heart disease and psychiatric illness were identified as a particularly vulnerable group (Naughton, et al., 2002).
- **France (2003).** Besides the high number of deaths in older people there were also much higher deaths in men of working age. (Toulemon & Barbieri, 2006).
- **Italy (2003)**. Increase in heart disease (Martiello, et al., 2005).
- **China (2010)**. CVA (stroke) was identified to increase during the heat waves (Chen, et al., 2015).
- **India (2015)**. People from low socio-economic groups were particularly vulnerable, presumably because of substandard housing or pre-existing untreated medical conditions (Zell, et al., 2015).

A complication in studies of heat stress is the potential time lag between exposure and development of symptoms. The majority of acute heat illnesses develop within 24 hours of exposure (Lavigne, et al., 2014), though the more chronic conditions resulting from heat stress may prevail for months (O'Brien, et al., 2012). However there appears to be a significant harvesting effect with hospital admissions being lower several days after an acute heat event. (Schwartz, et al. , 2004).

Impacts of workplace heat on different ill health categories

Illnesses or conditions that make a person more vulnerable to the effects of heat strain include a variety of diagnoses as described earlier. Our review of vulnerability indicates that people at risk include those with diseases of the heart, lungs, kidneys, diabetes/obesity, skin, nervous system, etc, as shown below. Users of drugs such as alcohol and amphetamines, older people and pregnant women are also at increased risk. We will discuss each of these conditions below.

Cardiovascular diseases

A review of the impacts of climate change on cardiac health (de Blois et al., 2015) analyzed the mechanisms by which heat conditions and other climate factors can affect heart disease. Heart strain is likely due to the relatively rapid changes in heart rate and blood pressure that heat exposure may cause. Experimental studies in physiological laboratories as well as epidemiological studies have found ample evidence for cardiac morbidity as well as mortality caused by excessive heat exposure. We will describe the main mechanisms in brief, while the published evidence is very extensive. Our focus below will also be on specific heat impacts on the health and well-being of people with existing cardiovascular diseases.

The mortality rate during the 2003 European heat wave was 30 % higher among people with heart disease than during an equivalent non-heat wave period (Hoffmann, et al., 2008). People with heart disease will respond differently to heat stress depending on the condition and the medications they take. For example, someone with congestive heart failure has a compromised output (from defective valves or a previous myocardial infarction). As the blood pressure drops when the skin blood vessels vasodilate, the heart is not able to maintain the core blood pressure (Cui & Sinoway, 2014). The use of beta blockers (Cui, et al., 2005) aggravates this situation as it prevents an increase in heart rate. Diuretics are commonly used to treat congestive heart failure and these can lead to dehydration symptoms and a further drop in blood pressure, so exertional heat strain symptoms develop (Casa et al., 2012). It has been shown (Engel, 1990) that skin blood vessel dilation is impaired in congestive heart failure. Then there is less blood flow to the skin (an apparent maximum of 4 L/min), and there is less ability to transport heat from the core so the core temperature rises and the symptoms of classic heat stroke develop. There is no evidence to show that sweating is impaired in congestive heart failure (Engel, 1990).

Whichever mechanism of heat strain, it is clear that people with *congestive heart failure* are more susceptible to heat stress. Kenny et al. (2010) reviewed the relative mortality risks of different health conditions during heat waves. Cardiovascular disease appeared as the highest increased risk (Naughton, et al., 2002; Bretin, et al., 2003). One study (Wilker, et al., 2012) found that the higher the heat stress the higher the levels of inflammation in people being treated for congestive heart failure. This higher level of inflammation increases the damage to cardiac muscle further reducing the ability of the heart to take thermoregulatory action.

Hypertension: This is a condition where there is an increased peripheral resistance to blood flow, for example because of atherosclerosis (clogged arteries), which could reduce the blood flow to the skin impairing thermoregulation (Kenny, et al., 2010). However, another study has shown that thermoregulatory

response to heat exposure may be unchanged by hypertension (Kellogg, et al., 1998).

Cerebro-Vascular Accidents (CVA) or Stroke: As described earlier, heat stroke can increase the incidence of disseminated intravascular coagulation, and studies of the London heat wave showed a near doubling of deaths due to cerebral and coronary thromboses (Keating, et al., 1986). One more recent study found that the incidence of CVA during a Chinese heat wave in 2010 (Chen, et al., 2015) was higher in rural areas than in urban areas of Nanjing province, in spite of the fact that urban areas are usually hotter than surrounding rural areas (from the urban heat island effect; Rizwan et al., 2008). The authors concluded that people in rural areas are more vulnerable to CVA deaths during episodes of high heat. This could be because of inferior medical services in rural areas, but it could also be because of poorer insulation of houses, harder work (in the heat) in rural areas and the higher possibility of dehydration because of less access to clean water (Chen et al., 2015).

Cardiac arrhythmias such as atrial fibrillation have in some studies been shown to increase with increasing heat (Michałkiewicz, et al., 2006). However, other studies have shown that this may be related to air pollution (Link, et al., 2013), which increases in hot weather. A study in 2008 showed no increase in cardiac arrhythmias during hot conditions, but some increase during cold conditions. (Głuszak, et al., 2008).

Respiratory diseases

Heat stress has a high impact on people with respiratory disease in some studies, with the risk of hospital admission being the third highest in studies of heat stress (Semenza, et al., 1996). The main respiratory condition predisposing people to heat strain was emphysema, with no increased risk for asthma and chronic obstructive pulmonary disease. Other studies show a lower risk (Kenny, et al., 2010). The mechanism for an increased susceptibility to heat stress for people with respiratory disease is uncertain, and it could be due to the increased level of air pollutants that accompany high temperatures (Kenny, et al., 2010).

In another study (Michelozzi et al., 2009) hospital admissions for respiretory and cardiovascular causes in association with heat waves in 12 European cities were studied, and the only statistically significant increase in admissions was for respiratory diseases. In California during a heat wave in 2006 (Knowlton et al., 2009) a small increased hospital admission rate for respiratory diseases was not statistically significant, and the greatest increase was seen for kidney diseases. On the other hand, reports on exercise induced asthma in athletes (Boulet & O'Byrne, 2015) indicate that temperature is a potential trigger for asthma attacks. Other textbook type information on the internet

indicates that cool temperatures are more common triggers than high temperatures. A sensitivity to heat among workers with respiratory diseases is not clearly established.

Kidney diseases

The physiological reactions to heat exposure involving redistribution of the blood to the skin, changed blood pressure and sweat related dehydration will all influence aspects of the kidney and urinary systems. People with existing kidney disease or related electrolyte imbalances will therefore be at risk. This is indicated by the especially increase rate of emergency department visits and hospital admissions during the heat wave in California in 2006 (Knowlton et al., 2009). Similar results were found in a study in Australia (Hansen et al., 2008). A study of many thousand young working people in Thailand (Tawatsupa et al., 2012) indicated that when working conditions were reported as hot, there was an increase reported prevalence of kidney disease. Thus, overheating and dehydration can be considered special risks for people with kidney diseases.

A major scientific discussion is continuing concerning a serious and often fatal chronic kidney disease epidemic in Central America (Wesseling et al., 2016). This disease occurs primarily in sugar cane workers with very intensive work in very hot conditions. They may sweat 10-15 L per day but only drink a few L of water during work. A similar disease problem occurs in Sri Lanka, but the cause is intensely debated. Some scientists believe it is caused by toxic agrochemicals, and more studies are ongoing. In any case, if this kidney disease is associated with daily dehydration, it is a sign of the specific sensitivity to heat that is likely to occur among kidney disease patients. Such patients may well be carrying out daily work, as long as they get dialysis.

Clinical data suggest that the heat related kidney disease in Central America, Mesoamerican Nephropathy, which affects sugarcane workers is not only associated with water loss as a key element of dehydration, but is also linked to repeated losses of essential minerals (Wesseling et al., 2016). All causal features of this heat related kidney disease issue are still not clear.

Diabetes

People with diabetes have a greater risk of heat related illness during hot weather (Schwartz, 2005). Diabetes Mellitus is a condition of high blood glucose. If untreated it can cause destruction of capillaries (in eyes, kidneys, fingers and toes), cause damage to nerves (especially loss of sensation from legs) and atherosclerosis. *Type 1 diabetes* is due to insufficient insulin production by the pancreas and by its damage to blood vessels it can interfere with vasodilation

in the skin and so compromise thermoregulation. During moderate exercise there is no difference between the heat loss of diabetic subjects and normal controls (Kenny, et al., 2016). However high levels of physical activity (metabolic rate > 400 W) thermoregulation is compromised (Carter, et al., 2014), and such high workloads can be expected in certain work situations (e.g. agriculture, construction and mining).

Type 2 diabetes: this is the most common form of diabetes, and it predominantly occurs in overweight people, so there is the added issue of obesity. There are relatively few studies of the effect of heat on people with type 2 diabetes. This is surprising because there is a significant increase of this disease during recent decades and also a significant increase in studies of other effects of heat stress (Kenny, et al., 2016).

People with type 2 diabetes have up to a 56 % greater risk for hospitalization and/or mortality during a heat wave.(Semenza, et al., 1996). Type 2 diabetes is usually of gradual onset. People with type 2 diabetes tend to be less healthy, with concomitant conditions such as obesity, high blood lipids, hypertension and other cardiovascular conditions. They also have a more limited skin blood flow than normal (Kenny, et al., 2016), and the temperature when skin vasodilation begins is higher than normal (Yardley, et al., 2013). Their sweating response is blunted unless they have a high level of fitness (Kenny, et al., 2016).

Heart disease is much more common in adults with diabetes than in the general population (Laing et al., 2003) (American Diabetes Association, 2012), so the problems discussed under cardiovascular disease will apply to many people with diabetes. Older people with type 2 diabetes have a significantly reduced capacity for heat loss so will be exposed to an even greater risk of heat related illness (Yardley, et al., 2013).

Skin conditions

Skin conditions can impede both vasodilation and perspiration, but because of the unsightly nature of skin conditions the person may cover themselves with more clothes than would be advisable in hot conditions. Sunburn can impair sweat secretion and cause fever. A person with skin scarred by burns will have less sweat glands and the scar tissue may prevent good heat exchange with underlying capillaries. Miliaria rubra (or prickly heat, heat rash, sweat rash) is when sweat glands are blocked by dead epithelial tissue, dirt or bacteria. (Leon & Kenefick, 2011) The sweat cannot escape to the surface so is unable to evaporate. This will hinder heat loss, but can often be resolved with increased skin hygiene. A number of congenital skin conditions like the lack of sweat glands can influence thermoregulation, but these conditions are uncommon (Epstein, 1989).

Multiple sclerosis

Most people with the rare disease multiple sclerosis (60 % to 80 %) are heat intolerant (Romberg et al., 2012). The adverse effects of heat are experienced in a number of ways such as pain, visual disturbance, reduced ability to walk or deteriorating cognitive function. The most prominent effect is fatigue – a common and disabling symptom in multiple sclerosis (Marino, 2009).The heat intolerance can be brought about by either high ambient temperatures or by heat generated by physical activity (Davis, et al., 2010). An increase of the core temperature by 0.8 °C is sufficient to produce a decrease in the central motor neuron conduction time and cortical excitability, most likely as a result of slowed conduction in the demyelinated neurons (White, et al., 2008). It is interesting to note that the number of admissions to hospital of people with multiple sclerosis was not increased during the major French heat wave in 2003 (Tataru et al., 2003). This may be due to the debilitating effects of heat on fatigue, which prevents physical activity and forces people to seek out cooler places (with air conditioning). It should also be noted that multiple sclerosis is more prevalent in the more affluent temperate regions of the world where most of the time it is relatively cool (Romberg et al., 2012).

The sweating response is reduced in some people with multiple sclerosis. In a cycling test, approximately 50 % of multiple sclerosis subjects exhibited normal sweating within the 30 minutes time of the exercise, while the remainder showed abnormal sweating in at least one site on the body (Mulcare, et al., 2001). The abnormal sweat response appeared to be more closely correlated to the level of fitness rather than the level of disability. A confounding issue in this research was that many people with multiple sclerosis were not able to sustain a high enough work intensity to elicit the sweat response.

The abnormal sweat response may be due to impairments of neural control of sudomotor (sweat gland) pathways or by changes to the sweat glands themselves. Vasodilation in the skin in response to heat is not impaired and was even, at times, greater than normal (Davis, et al., 2010). A further factor is dehydration as people with multiple sclerosis often voluntarily restrict fluid intake because of bladder urgency and frequency of urination (Davis, et al., 2010)

Spinal cord injury

People with spinal cord injuries have no sympathetic control below the site of injury so heart rate change, skin vasodilation or sweating response is limited or non-existent in that region. If core temperature rises then only the region above the injury can respond appropriately (Hopman & Binkhorst, 1997). This will make the person sensitive to heat exposure, especially when working.

Malignant hyperthermia

Malignant hyperthermia is a rare genetic condition that causes intense muscle spasms during exposure to some anaesthetics in combination with certain other drugs. The intense contractions generate so much heat that the core temperature rises and if vigorous cooling is not initiated the person dies from heat stroke (Larach, et al., 2010). In about 5-10 % of people with malignant hyperthermia the same response is triggered by exercise, heat stress or emotional stress (Wallace, et al., 2007). As the genetic mutation for this abnormality of drug metabolism has been identified, it is suggested that screening be carried out on susceptible populations (e.g. military recruits; (Leon & Kenefick, 2011).

Infectious diseases

A common finding in heat related illness is that there is a high incidence for people who have a current infection that causes a fever (Carter, et al., 2005). The body controls the temperature from the hypothalamus which has a "set point" normally near 37 °C. This set point temperature is reset to higher values when the hypothalamus is exposed to endotoxins, such as lipo-polysaccharides, from bacteria. The rise in body temperature assists the immune system to overcome the infection (Leon & Kenefick, 2011). Unfortunately, because the body temperature is set to a higher value, there is less margin of safety with heat stress before organ damage occurs. This makes people with a fever more susceptible to heat related illnesses.

Mental health and psychiatric conditions

Psychiatric patients have normally twice the death rate of other people, but during a heat wave their death rate climbs to four times that of others (Bark, 1998). An interesting effect here is that psychiatric patients under 65 years of age have a higher heat related mortality than those over age 65 (Page, et al., 2012). Psychiatric patients take anticholinergic medications (see section under drugs) which blunt the thermoregulatory response, and heat related mortality was high in the 1970s when high doses of this medication was used. However, even before the introduction of antipsychotic medication, psychiatric patients were at a higher risk of dying from heat related illness (Bark, 1998). The exact mechanism that puts psychiatric patients at risk is uncertain, but could include poverty, lack of air conditioning, living alone, and alcoholism. Wandering behaviour (in the heat) and agitation (increased muscle activity) could also increase the risk. Death by suicide is associated with higher temperatures (Page et al., 2007), and suicide is more common in psychiatric patients. Similar increases have been reported from Australia (Berry et al., 2010; Horton et al., 2010) and Asia (Kim et al., 2015). It should also be noted that violent crimes

increase in incidence during heat waves (Gamble & Hess, 2012; Raleigh et al., 2014) and this can be interpreted as a heat effect on psychologically vulnerable people.

Psychogenic fever

This is a rare condition where psychological stress causes a significant rise in core temperature (up to 41 °C) and as there is very little margin of safety between this fever and heat stroke, these people are very susceptible to heat illness (Oka, 2015). Many people will experience a small transient (about 1 hour) increase in core temperature (0.3 °C) when psychologically stressed. However, in some people this increase in temperature becomes chronic and can last for years, especially if subjected to continuous stress. In others the increase in temperature is much more significant, and these people rapidly develop a core temperature up to 41 °C (Oka, 2015). This fever is resistant to aspirin and NSAID because it is not caused by high levels of prostaglandins. Instead the stress directly effects the hypothalamus increasing its set point. People with this condition seem to have an increased sympathetic response and it has been shown that relaxation techniques and therapy to resolve suppressed negative emotions can be helpful (Oka, 2015).

People on drugs and medications

Thermoregulation involves the nervous system (hypothalamus, sympathetic nervous system), hormones (antidiuretic, aldosterone) and the cardiovascular system (heart, blood vessel dilation) so it is no wonder that many medications and drugs affect thermoregulation (Table 3) (Vanakoski & Seppala, 1998). As discussed earlier, drugs used in heart disease like beta blockers and diuretics compromise increased heart output and increased blood flow to the skin (Table 3). Many psychotropic drugs influence the autonomic nervous system and the hypothalamus and are linked to a wide variety of thermoregulatory problems (Table 3). Page et al (2012) found a significant link between antipsychotic (7 % increased risk), hypnotic (8 % increased risk) and anxiolytics (11 % increase risk) medications and heat related deaths. Indeed these authors suggest that the

Table 3. Medications and the mechanisms by which they increase the risks of heat strain. (Source: modified from WHO, 2011)

Medication	Mechanism
Anticholinergics (eg atropine)	Can affect central thermoregulation, reduce cognitive alertness and prevent or reduce sweating. Many of the drugs below have anticholinergic effects.
Antipsychotics	Can inhibit the sweating mechanism and reduce systolic blood pressure, central thermoregulation, cognitive alertness and vasodilation.
Antihistamines	Can inhibit the sweating mechanism and reduce systolic blood pressure.
Anti-Parkinson's drugs	Can inhibit the sweating mechanism, reduce systolic blood pressure and cause dizziness and confusion.
Tricyclic antidepressants	Reduces sweating; some can decrease centrally induced thermoregulation and cognitive alertness.
SSRI (eg Prozac)	Increases serotonin levels which can increase body temperature in high doses. (Liechti, 2014)
Anxiolytics and muscle relaxants	Reduce sweating and increase dizziness, decrease cardiac output and therefore reduce cooling by vasodilation, and worsen respiratory symptoms
Antiadrenergics and beta-blockers (eg propranolol)	Can prevent an increase in heart rate hence allowing a drop in blood pressure as the skin blood vessels vasodilate.
Sympathomimetics	Vasodilators, including nitrates and calcium channel blockers, can worsen hypotension in vulnerable patients
Antihypertensives and diuretics	Can lead to dehydration and reduce blood pressure; hyponatremia is a common side effect and can be worsened by excess fluid intake.
Antiepileptics	Can reduce cognitive alertness and increase dizziness.
Other drug classes such as antiemetics, anti-vertigo drugs, gastrointestinal drugs, urinary incontinence drugs	Also have anticholinergic effects.
Alcohol	Prevents the release of antidiuretic hormone so dehydration occurs due to excess urination.
Amphetamines (eg Ecstasy or MDMA)	Increases level of norepinephrine and serotonin which can cause hyperthermia (Sellers, et al., 1979), (Liechti, 2014)
Methyl Dopa, MAO inhibitors	Increases dopamine levels which are linked to hyperthermia (Liechti, 2014)
Cocaine	Increases level of norepinephrine and serotonin which can cause hyperthermia. (Liechti, 2014)

increase in heat related deaths of psychiatric patients during heat waves is almost entirely due to the medications they take (Page, et al., 2012). The

mechanism by which these medications stimulate the rise in core temperature is uncertain and is likely to depend on the drug. For example antipsychotics may alter the hypothalamus set point for thermoregulation (Kwok & Chan, 2005).

Alcoholism was one of the key causes of death in the Chicago heat wave of 1995 (Naughton, et al., 2002). Alcohol increases the blood flow to the skin so should be protective in that respect. However it also acts on the pituitary to decrease the level of ADH (anti-diuretic hormone) secretion which means more water is lost via the urine and dehydration results.

A drug that can cause a significant increase in core temperature, especially when it is already hot, is Amphetamine (e.g. MDMA also called Ecstasy) (Table 3). This has been extensively studied recently (Liechti, 2014). This drug stimulates the release of norepinephrine (noradrenalin), serotonin and to a lesser extent dopamine. The overall effect is raising core temperature by increasing body metabolism and reducing heat loss. The increase in body metabolism is most likely by MDMA's stimulation of norepinephrine which increases metabolism and causes vasoconstriction in the blood vessels of the skin. However it has been noted that both serotonin and dopamine also increase body temperature. (Liechti, 2014).

Effects of conditions related to health risks

Older age

It is clear that heat stress has a more significant effect as a person ages. Heat wave studies consistently report higher death rates among the elderly (Li, et al., 2015; Semenza, et al., 1996). Studies of Hong Kong construction workers show that older workers have a reduced *heat tolerance time* as the heat level rises (Chan, 2013). Workers in their twenties could cope with temperatures up to 31 °C for more than 119 minutes before they had to physically stop working. Older workers, in their fifties could only cope 56 minutes at 28 °C and 42 minutes at 31 °C. The definition of "heat tolerance time" varies depending on effect variable used, so comparisons between studies need special care.

Earlier research indicated that the reduction in heat tolerance in the elderly was caused by poor aerobic fitness and chronic health conditions rather than age (Pandolf, 1997). More recent research has shown that the ability to maintain core body temperature during heat stress is compromised in the elderly (Inbar, et al., 2004), but this is largely reversible by acclimatisation or increase of the level of aerobic fitness (Stapleton, et al., 2015a).

The reduced ability to lose heat from the body can lead to a 150 % increase in stored heat in the elderly compared to younger adults (Stapleton, et al., 2015a). This is due to a number of factors:

Firstly, the cardiac reserve is reduced in older people, possible due to a reduced sympathetic response (Blatteis, 2012).

Secondly, reflex vasodilation in the skin in response to heat is delayed and does not reach the maximum capacity of a younger person (Holowatz, et al., 2007, 2010). This could be due to the effect of aging on the cardiovascular system and a reduced level of hydration – a frequent condition of older people because their reduced sense of thirst and the inconvenience of incontinence. (Blatteis, 2012).

Thirdly, with aging the sweating response decreases especially in the lower limbs and the back (Inoue, et al., 2004). This reduction is from decreased individual sweat gland output and also a decrease in the number of active sweat glands (Blatteis, 2012). Stapleton, et al., (2015b) showed that the maximum heat loss by evaporation was less (about 10 % less) in an older female than a younger counterpart, and that this difference became greater as the work intensity increased. Part of the difference was explained by higher threshold for onset of sweating and also a decrease in thermo-sensitivity.

Thermal sensitivity also decreases with age and a few studies of behavioural changes have suggested that the elderly are less competent in protecting themselves from heat than younger adults (Guergova & Dufour, 2011). In addition, heat illness is often observed in older adults as a result of medications such as diuretics (Kenny, et al., 2010).

Obesity

Obesity, which is a common problem with Diabetes type 2, has its own risks during heat stress. Fatal heatstroke is 3.5 times more common in overweight adults than in people with average body weight (Henschel, 1967). Obese people have to work harder to walk or carry out weight bearing activities so they generate more heat than a non-obese person doing the same activity. There is less heat lost in an obese person because the area (where heat is lost) to volume (where heat is generated) ratio is less than for a slim person. When more body heat is generated than lost, core temperature rises. The rate at which it rises depends on the "specific heat capacity" (heat amount needed to be added to an object to increase its temperature by 1 degree). Adipose tissue has a much lower specific heat capacity than muscle or blood. So the body temperature of an obese person will rise more quickly than a non-obese person of the same weight.

Pregnancy and reproduction

Pregnancy is a period of particular health risks for the woman and her foetus. Environmental heat exposure is one hazard and it also affects the newborn and young child (Rylander et al., 2013). Pregnant women may suffer from increased body temperature due to hormonal changes during the pregnancy (Cunningham et al., 2010). Heat stress during reproduction is a key health hazard in many mammalian species (Hansen, 2009), and this applies to the male as well as the female. In the male, internal body heat levels affect the sperm parameters and reduce fertility (Sheiner et al., 2003). Another health risk related to pregnancy is the potential teratogenic impact of raised core body temperature (Edwards et al., 1995). These effects on pregnant women may occur at work, especially if the work is physically intensive causing major heat contributions from inside the body.

The birth-weight of newborn children is reported to be reduced by late pregnancy heat levels in the environment (Ha, et al., 2017), but the mechanisms are not clear. This study of more than 200,000 newborn children in the USA also showed birth-weight reductions during specific periods of the pregnancy when it was cold. This is similar to the findings in a review by Strand et al. (2011) which found increases of preterm birth, stillbirth, and low birth weight during the hot or cold seasons. Other aspects of children's health and well-being were also discussed by Rylander et al. (2013), but the links to the heat effects on a working pregnant woman is more distant.

Previous heat stroke

There is strong evidence that heat stroke victims are more susceptible to contracting heat illness again (Epstein, 1989). While residual effects may last up to 12 weeks, after that time the more susceptible people were just as susceptible as before the first exposure. There is a lag time of one day when people are exposed to high temperature and when they are hospitalised. If full symptoms have not developed when people go to work the next day, then the risks may be compounded. Wallace (2003) contends that there is a cumulative effect over two days only.

It has also been reported that 10 % of people have more than one episode of heat illness in one season (Wallace, 2003) and the speculation is that continual exposure presents an increased risk. Studies have shown (Shapiro & Seidman, 1990); (Shvartz, et al., 1977) that there is an increased vulnerability for several months following exertional heat stroke, while others have reported no added risk (Phinney, et al., 2001).

Many patients discharged from hospital after treatment for heat stroke continued to experience organ dysfunction during the following years (Leon & Kenefick, 2011). Following the 2003 heat wave in France the mortality rate

continued to climb from 58 % to 71 % two years after the event (Leon & Kenefick, 2011).

Other

There is likely to be other health situations or health related conditions that influence the sensitivity to heat of the working person. Further descriptive research on health issues related to heat will become more and more important as climate change progresses.

Research needs

The basic principles of heat physiology are well known, but there is a lack of research that quantifies the variation in heat sensitivity within different populations, and particularly the sensitivity of people with existing illnesses.

New research should identify and fill gaps in current knowledge in order to improve the quality of future health risk assessments based on data from models estimating future climate conditions.

There is an urgent need for health impact assessments of climate change impacts on heat exposures in workplaces and estimates of how people with special sensitivities can cope. Research ideas linked to the topic of this report were recommended in a review report for the Swedish Institute of Environmental Medicine (Kjellstrom, 2009), but unfortunately little follow-up of this was carried out in Sweden.

Conclusions

This report finds a clear physiological basis for special sensitivities to heat exposure in certain groups of people with existing ill health problems. External heat exposure as well as internal heat production due to muscular activities are both implicated in individual heat stress. In addition, clothing affects body heat management, and the combined impact of several factors determines the actual heat stress.

Heat stress leads to heat strain which can have serious clinical consequences. Core body temperature and sweating are key aspects of physiological responses to excessive heat exposure. The control of these variables into healthy ranges involves heart rate, blood flow in skin and internal organs, and sweating mechanisms. Any existing disease that has impacts on these functions can increase the heat sensitivity of the victim.

Diseases of concern include: Cardiovascular, Respiratory and Kidney diseases, Diabetes and obesity, Skin conditions, Multiple sclerosis, Spinal cord injury, Mental health and psychiatric conditions, and a few other conditions. In addition, health related characteristics such as old age and pregnancy, are linked to certain heat related risks.

As climate change progresses, excessive heat exposures on working people with existing illnesses is likely to become an increasing public and occupational health problem. Most people at risk will be living and working in tropical or sub-tropical areas, but even in Sweden one can expect future heat waves so hot that many people will suffer. Research and analysis on this problem is urgently needed.

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Redaktörernas slutord

Arbete i värme kan för friska människor innebär alltifrån försämrad komfort till värmeslag (heat chock), det senare är ett potentiellt dödligt tillstånd. För en människa som är sjuk innebär naturligtvis värmebelastning också samma risker, men ofta vid lägre värmebelastning. Dessutom kan det finnas speciella problem vid vissa sjukdomar. Den här översikten visar dels på allmänna principer för att förebygga skador vid värmebelastning som kan tillämpas på alla med sjukdom, men också för friska. Den visar också på att det saknas studier av sjuka som utsatts för värmebelastning i arbetet. Sådana studier torde i många fall vara svåra att genomföra, så att man i praktiken måste tillämpa mer generella kunskaper. Översikten visar också att ökande besvär, liksom risk för försämring, kan förväntas vid många olika sjukdomstillstånd.

Från värmeböljor har man bland annat kunnat visa en ökad dödlighet i anslutning till värmeböljan, till exempel i Frankrike 2003. Huvudsakligen är det en ökning bland redan sjuka människor varav många inte är i arbetslivet. I Sverige har denna kunskap lett till att man utarbetat vårdprogram/handlingsplaner som kan tillämpas inom hemvård och sjukvård. Några motsvarande svenska vårdprogram riktade mot sjuka personer i arbetslivet känner vi inte till.

Vid arbete i varma länder finns möjligheter till acklimatisering vilken vanligen tar ca 10 dagar. Värmeböljor i Sverige är oftast så pass kortvariga att man inte kan påräkna någon acklimatisering och framförhållningen är också ganska kortvarig. Via väderlekstjänsten vet man endast med någorlunda säkerhet om det blir en värmebölja ett fåtal dagar i förväg.

När man ska bedöma om man måste vidta särskilda åtgärder när en sjuk person utsätts för värmebelastning i arbetet har sjukdomens svårighetsgrad stor betydelse. Arbete vid måttlig värmebelastning kan kanske vara möjlig för en person med lindrig hjärtsvikt medan samma arbetssituation med påtaglig hjärtsvikt kan innebära risk för en betydande försämring. Dessutom kan viss typ av läkemedelsbehandling, liksom hög ålder, öka känsligheten för skador vid värmebelastning. De flesta personer med svårare sjukdomar är både äldre och har läkemedel. Eftersom allt fler personer arbetar vid hög ålder och värmeböljor kan förväntas bli mer frekventa blir frågeställningen om arbete med sjukdom vanligare.

Gravida har också en ökad känslighet och i extrema fall kan fostret skadas. I översikten beskriver författarna en lång rad av sjukdomstillstånd som kan påverkas. Man konstaterar att det inte alls finns samma kunskap om risker för dessa personer jämfört den forskning som gjorts på personer som arbetar i varm miljö med tungt fysiskt arbete.

I dagens svenska arbetsliv torde det vara mindre vanligt med långvarigt tungt fysiskt arbete i värme för personer med kronisk sjukdom. Däremot kan

man förvänta sig episoder med ”värmeböljor” och om det då inte finns luftkonditionering kan också personer med ganska stillasittande arbete utsättas för en värmebelastning som kan påverka deras hälsa påtagligt. Eftersom en sådan värmebölja kommer med ganska kort framförhållning kan man inte vänta med att informera den sjuke eller arbetsledning om vad som ska göras tills värmeböljan kommer, utan sådan kunskapsuppbyggnad måste ske planerat, förslagsvis vid hälsokontroller inom företagshälsovård och skyddsronder. Informationen behöver anpassas både till värmebelastningen och sjukdomen. Ibland kan personen själv förändra arbetet och värmebelastningen men ibland är det omöjligt, till exempel i omvårdnad och många serviceyrken. Det gäller då att också arbetsledningen är införstådd med behovet av anpassning.

Det finns således ett behov att förebygga ohälsa hos personer med kronisk sjukdom som kan utsättas för värmebelastning i arbetet. Denna typ av bedömning blir individuell och innebär att läkaren har kännedom om både sjukdomens svårighetsgrad och värmebelastning liksom möjligheterna till anpassning av arbetet vid värmeböljor. Att förebygga risker handlar i många fall om att inta tillräckligt med vätska, om möjligt söka sig till svalare områden och minska på graden av fysisk ansträngning. I vissa fall kan tekniska lösningar, till exempel kylande kläder, vara motiverade.

För vissa sjukdomar, som hjärtsvikt, är det sannolikt väl känt för de flesta att värmebelastning kan påverka sjukdomen. Däremot är sannolikt mindre känt att man också kan krävas särskilda åtgärder för personer med mental ohälsa. Den begränsade forskning som finns visar att bland annat självmord är vanligare vid värmeböljor liksom att flera olika läkemedel som används vid mental ohälsa påverkar känsligheten för värme. Det finns därför anledning att ge råd till personer med mental ohälsa om risker vid värmebelastning, liksom till arbetsledning. Översikten visar också att man bör ta särskild hänsyn till personer med MS (multipel skleros) där många har en ökad värmekänslighet och försämrad svettfunktion.

Åtgärder för att förebygga ogynnsam värmebelastning hos sjuka och personer med läkemedelsbehandling kan också ha mer generella effekter på riskerna i arbetslivet. Översikten påpekar att olyckor är vanligare vid värmebelastning. En person som har ADHD och som behandlas med amfetamin kan vid värmebelastning försämras, få försämrad impulskontroll och utsätta sig själv och andra för en ökad olycksrisk (se dokumentet av Kjellström och Lemke).

Översikten visar dels på ett behov av ytterligare forskning men också på att även i land som Sverige behöver vi ta särskild hänsyn till värmebelastning i arbetet hos personer med sjukdom. Här torde företagshälsovården ha en viktig roll att fylla.

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